

Spinal epidural haematoma following removal of epidural catheter after an elective intra-abdominal surgery

INTRODUCTION

Although epidural catheters are effective for post-operative analgesia,^[1] they are not free of risks. A rare complication of catheter-based epidural analgesia is bleeding within the epidural space resulting in the formation of a spinal epidural haematoma (SEH) with neurological deficits. We encountered a case of epidural haematoma and lower limb paresis following removal of epidural catheter in post-operative period.

CASE REPORT

An 86-year-old female weighing 58 kg, with controlled hypertension underwent elective choledochoduodenostomy under general anaesthesia with endotracheal intubation. Before induction of general anaesthesia, an epidural catheter was inserted uneventfully at T7-8 intervertebral level using 18 gauge Tuohy needle with 20 Gauge catheter; epidural space was identified using loss of resistance to air in the first attempt, with no blood seen through epidural needle and catheter. A test dose of 3 ml 2% lignocaine with adrenaline 1:200,000 was used to test to exclude intravascular and intrathecal placement of epidural catheter. Pre-operative investigations were within normal limits including creatinine (0.97 mg/dl), prothrombin time, activated partial thromboplastin time and platelet count.

Post-operative analgesia was provided with continuous epidural infusion of bupivacaine 0.125% along with fentanyl 2 µg/ml at the rate of 4–8 ml/h. Mechanical thromboprophylaxis (pneumatic pump) was started pre-operatively with addition of a single daily dose of fractionated heparin (dalteparin sodium 2500 U) subcutaneously after 12 h post-operatively. Post-operative course was uneventful. On the 4th post-operative day, 12 h after the last dose of fractionated heparin, the epidural catheter was removed. Four hours after removal of the epidural catheter, the patient complained of severe backache with radiating pain and weakness

in the lower limbs. Clinical examination revealed hypoesthesia and muscle power of grade 3/5 in the left lower limb and 4/5 in the right lower limb. With a high index of suspicion for SEH causing spinal cord compression, the patient was started on intravenous (IV) methylprednisolone, and further dosage of heparin was withheld. A magnetic resonant imaging (MRI) scan was deferred after discussion with radiologist and surgeon as the patient had staples at skin incision site. Computed tomography thorax was done which revealed partial collapse of L1-3 vertebrae, and vague intraspinal soft tissue lesion in L1 extending to T11-L2 level. On the 6th post-operative day (48 h after symptoms were noted) after removing the skin staples, an MRI scan was done. MRI revealed epidural haematoma at T11–L1 level with cord compression with no significant cord lesion [Figures 1 and 2].

The patient was taken up for emergency laminectomy and decompression under general anaesthesia. The haematoma was meticulously evacuated, and no active bleeding vessel could be identified.



Figure 1: Magnetic resonant imaging thoracolumbar spine showing epidural haematoma compressing the spinal cord

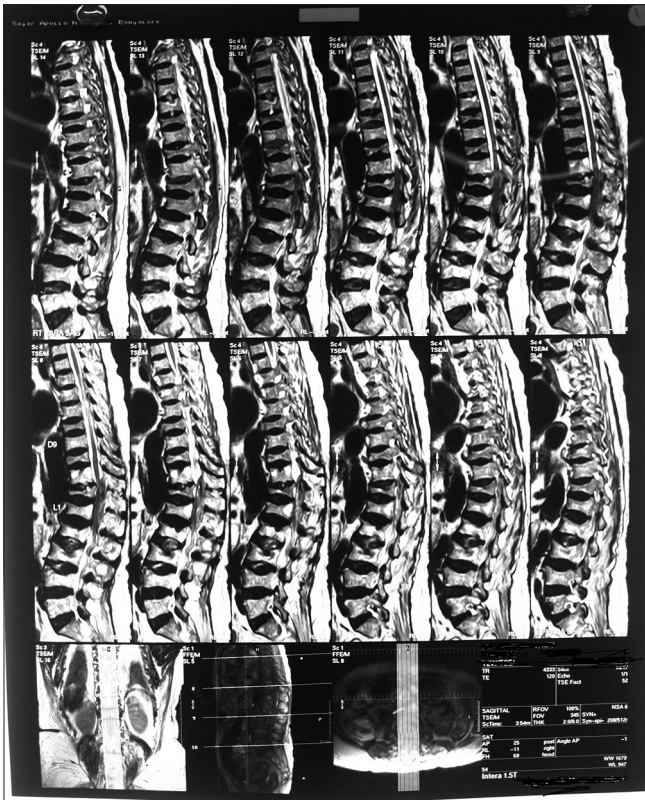


Figure 2: Magnetic resonant imaging showing extension of epidural haematoma to thoracic 11-lumbar 1 region with underlying cord compression

Her neurological status remained unchanged for first 2 days; however, the radiating pain had disappeared. On the 3rd post-operative day, improvement in muscle power was seen. On the 5th post-operative day, the patient regained normal muscle power of both lower limbs. She was discharged home on the 6th post-operative day; subsequent follow-up on weekly basis did not reveal any sensory-motor abnormality.

DISCUSSION

The incidence of bleeding complications has been previously estimated to occur in 1:150,000 epidurals and 1:220,000 spinal anaesthetics. However, it is highly variable and may be much higher (1:3000 epidurals and 1:40,000 spinal anaesthetics) with concomitant use of heparin.^[2-5]

The symptoms of acute SEH include sudden onset of back pain, often with a radicular character and sensory-motor deficits which outlast the expected duration of epidural or spinal anaesthesia. Symptoms of SEH may be masked by continuous infusion of epidural analgesia. The severity of motor or sensory deficit may be greater than expected when continuous epidural infusion is used.^[6-8]

MRI is the investigation of choice when SEH is suspected after an epidural placement or removal. In the present case, MRI could not be done as first line investigation, as the patient had skin staples. It was performed when deemed permissible, i.e., after removing the skin staples. The epidural needle insertion site was T7-8 space; the downward extension of haematoma in T11-L1 could be due to injury to any vessels by downward migration of epidural catheter during insertion or removal.^[7] The patient was put on methylprednisolone after the advice of neurosurgeon; treatment was initiated as per the protocol of traumatic spinal cord injury although the evidence for its use is poor and often questionable.^[8]

Once a definitive diagnosis has been made, urgent surgical decompression is the accepted standard of care in the vast majority of cases. This usually requires laminectomy and evacuation of the haematoma,^[6,7] which was carried out in our case. In the recent years, there have been several case reports of SEH, which have been followed conservatively, without surgical intervention and with favourable neurological outcomes.^[9,10] Most studies have found a clear trend of better outcomes in those patients who underwent operative intervention early (within 6 h of symptoms) and in those patients presenting with less severe deficits.^[10,11]

In our case, the time interval between symptom onset and surgery was more than 24 h, but still the patient had a satisfactory recovery. It has been previously shown experimentally that functional recovery in cats following spinal cord compression was associated with demyelination of the spinal cord white matter.^[11] Therefore, the patients who recover after surgery probably had a demyelinating lesion, whereas patient who does not recover may have axonal disruption.

Recent guideline from American Society of Regional Anesthesia recommend the minimum timing of epidural placement and removal as follows:^[12]

- 4 h after IV unfractionated heparin, can be restarted 2 h after placement/removal of catheter
- 8–10 h for subcutaneous heparin
- 12 h for subcutaneous fractionated heparin; can restart after 4 h of epidural placement/removal.

Development of epidural haematoma after removal of the catheter in our case may have been caused by the use of fractionated heparin (dalteparin 2500 units), although the time interval from the last dose of dalteparin to the removal of catheter was 12 h.

Although the serum creatinine seems to be in normal range (0.97 mg/dl), but its value may be misleading specially in elderly with reduced muscle mass where the creatinine production as such is very low and the estimated creatinine clearance by Cockcroft–Gault equation was just 39.3 ml/min. This might have prolonged the elimination of fractionated heparin, and sufficient level of anticoagulant activity was still present when the catheter was removed. This complication might have been averted if the catheter removal had been done after 24 h of the last dose of dalteparin or use of unfractionated heparin in the presence of reduced creatinine clearance which does not depend on renal clearance for elimination.

CONCLUSION

Occurrence of spinal epidural haematoma may be attributed to longer duration of action of fractionated heparin in elderly patients with impaired creatinine clearance. Delaying catheter removal for 24 rather than 12 h after the last dose of heparin may be the more appropriate.

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Conflicts of interest

There are no conflicts of interest.

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